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ART UNIT	PAPER NUMBER
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	09/718,355	ROULEAU ET AL.	
	Examiner	Art Unit	
	DANIEL KOLKER	1649	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 February 2008*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *42-55 and 60-64* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *42-55,60 and 61* is/are rejected.

7)☒ Claim(s) *62-64* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. The remarks and amendments filed 19 February 2008 have been entered. Claims 42 - 55 and 60 - 64 are pending and under examination.

Continued Examination Under 37 CFR 1.114

2. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 19 February 2008 has been entered.

Withdrawn Rejections and Objections

3. The following rejections set forth in the previous office action are withdrawn:

A. The rejection under 35 USC 102(b) over Rechkziegel is withdrawn in light of the amendments. The claims now require that the composition used in the claimed methods comprise a recombinantly expressed alpha subunit of a sodium channel. While the step of transfecting nucleic acid encoding the relevant channel into cells is not explicitly recited, it is implied. Rechkziegel teaches contacting test compounds with human cells that endogenously express the relevant sodium channel, but the reference does not teach contacting with compositions comprising recombinant sodium channels as claimed.

B. The rejections under 35 USC 103(a) as obvious over Rechkziegel are withdrawn in light of the amendments. As set forth in the previous paragraph, Rechkziegel does not anticipate the method of claim 42, as amended. The other cited references in the rejections under 35 USC 103(a) fail to make up for the deficiencies of Rechkziegel.

C. The rejections under 35 USC 112, second paragraph are moot as claims 56 – 58 have been canceled.

New Rejections

Priority

4. Applicant’s claim for the benefit of a prior-filed application under 35 U.S.C. 119(e) or under 35 U.S.C. 120, 121, or 365(c) is acknowledged. Applicant has not complied with one or

more conditions for receiving the benefit of an earlier filing date under 35 U.S.C. 119(e) as follows:

The later-filed application must be an application for a patent for an invention which is also disclosed in the prior application (the parent or original nonprovisional application or provisional application). The disclosure of the invention in the parent application and in the later-filed application must be sufficient to comply with the requirements of the first paragraph of 35 U.S.C. 112. See *Transco Products, Inc. v. Performance Contracting, Inc.*, 38 F.3d 551, 32 USPQ2d 1077 (Fed. Cir. 1994).

The disclosure of the prior-filed application, Application No. 60/167623, fails to provide adequate support or enablement in the manner provided by the first paragraph of 35 U.S.C. 112 for one or more claims of this application. The examiner is unable to find disclosure of the D188V mutation in SCN1A in the provisional application. While the D188V mutation is disclosed in the instant specification at p. 55 lines 3 - 25, the examiner is unable to find support for this limitation in the provisional application. This mutation is recited in claims 42 and 62, and is encompassed by claims 43 – 53, 60, and 61 as well. Therefore, for the purposes of applying prior art, the effective filing date of claims 42 – 53 and 60 – 62 is the date the instant application was filed, 24 November 2000. Claims 54 – 55 and 63 – 64 are entitled to benefit of the provisional application, so the effective filing dates of those claims is 26 November 1999, the date 60/167623 was filed.

Should applicant disagree with the examiner's factual determination, applicant should provide evidence or point to evidence currently of record which indicates that the provisional application provides support for the limitations recited in claim 42 part (iv) and claim 62. This could be accomplished, for example, by pointing to specific page and line numbers or to figures or sequences in the 60/167623 application.

Claim Rejections - 35 USC § 102

5. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 42 – 47, 49 – 51, 53, 60 – 61 are rejected under 35 U.S.C. 102(b) as being anticipated by Noda (1987. Journal of Receptor Research 7:467-497, cited in office action mailed 16 April 2007).

A similar rejection was made in the office action mailed 16 April 2007, see paragraph number 10 on page 7. The rejection was withdrawn in the office action mailed 29 October 2007, as the claims at that point required that the protein used in the assay be identical to SEQ ID NO:3 or 4, or encoded by SEQ ID NO:1 or 2. Since the claim amendments filed 19 February 2008 expand the scope of the claimed invention to include screening methods wherein the relevant channel is encoded by a nucleic acid at least 95% identical to SEQ ID NO:1 or 2, the rejection for anticipation by Noda is reinstated.

Noda teaches rat sodium channels which are 2009 amino acids long and are 98.5% identical to applicant's SEQ ID NO:4 (see sequence alignment mailed with office action on 16 April 2007, see also Noda p. 469 final paragraph and Figure 2). The proteins are thus within the scope of claim 42, particularly part iii, as the proteins are encoded by nucleic acids (see Noda, Figure 2), and the specification discloses that SEQ ID NO:2 encodes SEQ ID NO:4. Thus it is apparent that the protein from Noda is encoded by a sequence at least 95% identical to SEQ ID NO:2. Noda teaches contacting cells carrying the nucleic acids encoding this protein with agents; see p. 469 for identification of cells carrying the nucleic acids in vectors (and therefore on point to claims 46 – 47) as well as p. 485 which indicates that cells expressing the vectors were contacted with TTX. Note that Noda teaches the percentage of the current which is TTX-sensitive and therefore it is reasonable that the authors measured the amount of sodium channel activity in both the presence and absence of TTX, as encompassed by claim 42. See in particular Noda, p. 482, Figure 6, which shows the currents through the sodium channel before and after application of TTX. Noda teaches that TTX prevents sodium channel activity, and therefore is on point to element (d) of claim 42. While Noda refers to the sodium channel used as "sodium channel II", not "SCN1A" as recited in the claims, since the prior art sequence meets the structural limitations of claim 42 part (iii), it must in fact be a "SCN1A protein having sodium channel activity" as recited in the claim. Thus the reference by Noda anticipates every element of claim 42.

Claims 43 – 44 and 53 are rejected as they differ from claim 42 only in the intended use of the method, which is not given patentable weight; claims 43 – 44 and 53 require no additional steps or materials beyond what is recited in claim 42. Claim 45 is rejected because TTX is

contained in a library consisting of at least TTX and saxiotoxin (STX); see Noda, p. 479 first complete paragraph, which indicates that both these compounds were tested. There is no explicit definition of a "library", but it is reasonable that this term encompasses more than a single compound. Claim 49 is rejected as the cells used in the assay are whole cells (*Xenopus* oocytes). Claim 50 is rejected as the reference by Noda reports that changes in currents are measured, as is flux of ions through the channel, as current is in fact flux of ions through a channel. Claim 51 is anticipated as Noda used two-electrode voltage clamp to measure flux of ions through the channel; see p. 469, last sentence of paragraph entitled "Materials and Methods". Claim 60 is anticipated as the cells used by Noda are reasonably indicator cells. The term "indicator cells" is defined at p. 21 final paragraph of the specification as "cells that express at leas one sodium channel α subunit according to the present invention" and which can be used in screening assays. Claim 61 is anticipated as the protein from Noda is over 98% identical to instant SEQ ID NO:3.

Claim Rejections - 35 USC § 103

6. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 42 – 51, and 53, and 60 – 61 are rejected under 35 U.S.C. 103(a) as being unpatentable over Noda (1987. Journal of Receptor Research 7:467-497) in view of Hartshorne

(1984. Journal of Biological Chemistry 259:1667 – 1675, cited in office action mailed 16 April 2007).

The reasons why claims 42 – 47, 49 – 51, 53, and 60 – 61 are anticipated by Noda are set forth above. The reference teaches screening sodium channels for antagonists which bind to the channels including STX and TTX, but does not teach cell-free assays as recited in claim 48.

Hartshorne teaches purifying sodium channels to homogeneity from tissue and teaches that the purified channels are able to bind STX. The reference teaches that the isolation process increases the concentration of sodium channels over 1300-fold. However Hartshorne does not explicitly teach screening methods as recited in claim 42.

It would have been obvious to one of ordinary skill in the art to use the cell-free binding assays described by Hartshorne in the screening assays of Noda, with a reasonable expectation of success. The artisan of ordinary skill would be motivated to use the purified channels and cell-free binding assay, because doing so would allow the artisan to use considerably less of the agents to be screened, thereby reducing costs. Furthermore, the artisan of ordinary skill would be motivated to use *in vitro* binding assays as both references teach that those agents which bind to the STX binding site block the channel activity, thus screening for binding to this region would identify sodium channel blockers. The *in vitro* cell-free binding assay would be advantageous, because it can easily be scaled up to allow for screening of many agents, whereas the scaling up an *in vivo* assay such as that described by Noda would be difficult and expensive, given the high cost of electrophysiological recording equipment. This would have been immediately obvious to the artisan upon reading the references, as the Hartshorne reference teaches that STX-binding activity is retained, and the substantial increase in purity would mean that much less of a candidate compound would be needed in a given reaction volume.

7. Claims 42 – 47, 49 – 53, and 60 – 61 are rejected under 35 U.S.C. 103(a) as being unpatentable over Noda (1987. Journal of Receptor Research 7:467-497) in view of Kienle (1997. Biosensors and Bioelectronics 12:779-786, cited in office action mailed 16 April 2007).

The reasons why claims 42 – 47, 49 – 51, 53, and 60 – 61 are anticipated by Noda are set forth above. The reference teaches screening sodium channels for antagonists including

STX and TTX, but does not teach using surface plasmon resonance, as recited in claim 52, to detect the interaction between the putative antagonists and the channel.

Kienle teaches use of surface plasmon resonance, as recited in claim 52, to identify agents which bind to rat cardiac sodium channels, which are functionally similar to the sodium channels in claim 42. The reference by Kienle teaches that surface plasmon resonance is a powerful tool that offers many advantages in identifying interactions between a ligand (for example, an antagonist) and a receptor (i.e. a channel). These advantages are listed on p. 779 second column and include that the technique allows for label-free detection, and that it is direct and non-invasive. Further the technique allows for measurement of kinetic rate constants and affinity constants, both of which are useful for the artisan to know when developing drugs. However, Kienle does not teach screening assays using the specific sodium channels recited in claim 42.

It would have been obvious to one of ordinary skill in the art to modify the assays of Noda, which screen for antagonists that bind to sodium channels, to include the surface plasmon resonance technique taught by Kienle, with a reasonable expectation of success. The motivation to modify the assays would be to take advantage of the addition information provided by the surface plasmon resonance technique, including kinetic rate and affinity constants.

8. Claims 42 – 47, 49 – 51, 53 – 54, and 60 – 61 are rejected under 35 U.S.C. 103(a) as being unpatentable over Noda (1987. Journal of Receptor Research 7:467-497) in view of Wood (WO 97/01577, published 16 January 1997), Malo (1994. Cytogenet Cell Genet 67:178-186, cited as reference C2 on IDS filed 28 March 2003), and Current Protocols in Molecular Biology (1989 – 1996, pages 6.0.3 – 6.0.5, 6.1.1 – 6.1.4, 6.3.1 – 6.3.6, and 6.5.1 – 6.5.2).

The reasons why claims 42 – 47, 49 – 51, 53, and 60 – 61 are anticipated by Noda are set forth in the rejection under 35 USC 102(b) above. Briefly, Noda teaches screening sodium channels for antagonists which bind to the channels including STX and TTX. Noda teaches recombinant cells comprising rat sodium channels, which are within the scope of claim 42, particularly element (iii), as well as claim 61. However Noda teaches the rat sodium channel, and does not specifically teach methods of using a sodium channel protein with the sequence of SEQ ID NO:3, encompassed by claims 42 and 54. SEQ ID NO:3 is the human adult form of human sodium channel 1A (specification, p. 27, lines 16 – 17).

Wood teaches a number of screening methods with recombinant sodium channels, and teaches that they are useful for identifying new drugs. See p. 2 line 18 – p. 3 line 2, p. 3 line 30 – p. 4 line 2, and p. 38 line 3 – p. 39 line 17. The methods are on point to claims 42 - 44 (note claims 43 – 44 recite an intended use of the assay), 49 (the assays are performed in cells expressing the nucleic acid; p. 38 line 21 – 22), 50 – 51 (note radioactive guanidine is to be used to measure ion flux; p. 39 first complete paragraph), 53 (which recites intended use of the assay), and 60 (the cells are indicator cells). Wood also teaches that when a sodium channel from one species is known, and nucleic acid encoding the channel is in hand, one can screen a cDNA library made from a second species in order to obtain the nucleic acid encoding the same protein from the second species. Note that Wood indicates while certain examples discuss rat sequences, human sequences can also be used (p. 5 lines 18 - 31 for example). Wood also teaches alternative methods to identify human sodium channel sequences, by using PCR, and teaches how to confirm that the appropriate clone has been obtained with an *in vitro* assay (p. 28 line 18 – p. 32 line 2). However while Wood teaches these methods relating to sodium channels expressed in the periphery, the reference does not explicitly teach sodium channels encoded by nucleic acids at least 95% identical to SEQ ID NO:3, as encompassed by claims 42, 54, and 61.

Malo teaches nucleic acids which are partial sequences of human sodium channel 1 alpha, also known as SCN1A. See in particular p. 179 second column ("Results"), Figure 2 on p. 181, p. 182 "Assignment of a human brain sodium channel 1α (SCN1A) gene", and Figure 3, appearing on p. 183, which shows the sequence of the isolated nucleic acid and encoded protein. Note that Figure 3 shows substantial identity between the human SCN1A sequence and that rat Scn1a (also called RBI) sequence at the amino acid and nucleic acid levels. While the human sequence is obviously only a partial sequence, at both the nucleic acid and amino acid levels, the high degree of homology between the identified human sequences and the rat sequence known in the prior art (Note that Figure 3 from Malo indicates that the rat sequence is that from Noda 1986; this is the same sequence used by Noda 1987, cited above; see also Noda 1987 p. 469 and 496) suggests to the artisan of ordinary skill that the full-length human sequence could be obtained. Malo teaches that the partial sequence was obtained by PCR on a human genomic library (p. 179 first column). However Malo does not teach the full-length sequence of SEQ ID NO:3, or nucleic acids encoding the full-length sequence, and does not teach the screening assays recited in claim 42.

Current Protocols Chapter 6 excerpts teaches the artisan of ordinary skill how to screen a DNA library to obtain clones. The specific techniques and protocols necessary for the experiments are detailed, and troubleshooting tips are presented. Chapter 6.1 teaches the artisan of ordinary skill how to plate libraries and transfer them to filters; Chapter 6.3 teaches how to hybridize a DNA probe to the filters, and Chapter 6.5 provides guidance in isolating the appropriate clone. Note that each of the individual articles was originally published between 1989 and 1996, as indicated by the dates on the face page of each chapter. Thus the reference describes techniques to screen libraries and pull out a full-length clone which were well–known prior to the effective filing date of this invention. However Current Protocols does not teach either sodium channels or screening assays.

At the time the invention was made, it would have been obvious to one of ordinary skill in the art to screen a human DNA library in order to obtain a full-length clone encoding SCN1A and to use such a nucleic acid in the screening assays described by Noda, with a reasonable expectation of success. The motivation to do so would be to find inhibitors of human sodium channels, instead of rat sodium channels used by Noda. This motivation comes directly from the references themselves; note that Malo teaches that human tissues express SCN1A-encoding nucleic acid, and teaches that such nucleic acids can be obtained by screening libraries. The artisan of ordinary skill would have a reasonable expectation of success in obtaining full-length nucleic acid encoding human SCN1A (i.e., encoding the protein of SEQ ID NO:3), given that Noda provides the full-length rat sequence and Malo provides a partial human sequence. Either of these could be used as a probe in screening a library, such as the commercially available libraries used by Malo, or by making cDNA libraries from human neural tissue as described by Wood (p. 23 lines 5 – 11). Note that the Current Protocols chapters provide guidance to the artisan of ordinary skill in library-screening procedures, and Wood indicates that such procedures are suitable for purifying nucleic acids encoding human sodium channels. Further Wood indicates that these types of nucleic acids can be used in screening assays. Following the guidance set forth in these references, the artisan of ordinary skill would arrive at the invention of claims 42 and 54, as human SCN1A is in fact the protein of SEQ ID NO:3. Isolation of the human, as opposed to the rat, SCN1A-encoding nucleic acid would not have been the result of innovation but rather of common sense. The reasons why claims 43 – 47, 49 – 51, 53, and 60 – 61 are included in this rejection are set forth in the rejection under 35 USC 102(b) above and for the sake of brevity will not be repeated here.

9. Claims 42 – 47, 49 – 51, 53 – 55, and 60 – 61 are rejected under 35 U.S.C. 103(a) as being unpatentable over Noda in view of Wood, Malo, and Current Protocols in Molecular Biology as applied to claims 42 – 47, 49 – 51, 53 – 54, and 60 – 61 above, and further in view of Avanzini (1996. Progressive Nature of Epileptogenesis (Epilepsy Res. Suppl. 12), pp. 53 – 61, of record) and Soares (U.S. Patent 5,482,845, issued 9 January 1996).

The reasons why claims 42 – 47, 49 – 51, 53 - 54, and 60 – 61 are obvious over Noda in view of Wood, Malo, and Current Protocols in Molecular Biology are set forth in the previous rejection. The references provide guidance and motivation to one of ordinary skill in the art to obtain the nucleic acid encoding SEQ ID NO:3, and to use it in a screening assay. However none of the references explicitly teach the neonatal form of human sodium channel 1A or nucleic acids encoding same, as encompassed by claim 55 (note SEQ ID NO:4 is the neonatal form of the channel; see specification p. 27 lines 17 – 18).

Avanzini teaches that there are several forms of epilepsy which are unique to human infants and which have onset at very early age (<1 yr., see Avanzini p. 53 first column). Avanzini teaches that there has been difficulty in reproducing the features of these early infantile forms of epilepsy in animal models, thereby indicating to the artisan of ordinary skill that there is a need to screen for agents which could treat epilepsy in humans. Avanzini teaches performing experiments with neural tissue from juvenile rats (see Methods section), and teaches that in juvenile tissue inhibitory post-synaptic potentials are not present (p. 53 second column), indicating neurons are likely to be hyper-excitable. Avanzini teaches contacting tissue comprising juvenile rat sodium channels with TTX (p. 54 second column). However Avanzini does not teach performing screening assays to find drugs which reduce sodium channel activity in recombinant neonatal sodium channels.

Soares teaches cDNA libraries from neonatal infant brain (column 4 line 66 – column 5 line 2; column 12 line 15 – column 15 line 44), which is on point to claim 42 part (ii) and claim 55, as SEQ ID NO:4 is the neonatal form of the sodium channel; see specification p. 27 lines 17 – 18. The reference provides detailed instructions on how to make such a library, and how to normalize it. However, Soares does not explicitly teach nucleic acids encoding sodium channels and does not teach screening assays as recited in claim 42.

It would have been obvious to one of ordinary skill in the art to screen the human neonatal brain cDNA library from Soares, obtain the neonatal form of the SCN1A channel, and

use it in the screening assays described by Noda and Wood, with a reasonable expectation of success. The motivation to do so would be to identify agents to treat epilepsy in juvenile humans, as Avanzini teaches that the animal models do not reproduce this disease well. Thus the artisan of ordinary skill would be motivated to follow the guidance in Wood, Malo, and Current Protocols to obtain the cDNA encoding human juvenile sodium channels, to use it in a screening assay. It would be reasonable to expect success, as Avanzini teaches that those manipulations which affect sodium currents (i.e., those which pass through sodium channels) are effective in blocking bursts of neural activity (pp. 57 – 58), which are associated with epilepsy.

Allowable Subject Matter

10. The prior art does not teach or suggest the three specific mutations recited in claim 42, parts (iv), (v), and (vi); these mutations are also recited in claims 62 - 64.

11. The art made of record and not relied upon is considered pertinent to applicant's disclosure: Wallace U.S. Patent 7,078,515. The reference teaches sodium channel 1A (SCN1A) and its isolation from humans; see for example column 15 line 50 – column 16 line 57. Wallace also teaches that the D188V mutation within this sequence leads to increased risk of epilepsy; see column 17 lines 13 – 32, as do several other mutations (column 17 lines 33 – 58). While Wallace teaches the full-length SCN1A protein (it is Wallace's SEQ ID NO:10; see column 20 lines 53 – 56), the protein is not identical to applicant's SEQ ID NO:3 or 4; applicant's sequences have E at residue 1455, whereas Wallace's SEQ ID NO:10 has a K. Additionally, the earliest effective U.S. Filing date of Wallace '515 patent is 20 December 2001, the date that PCT/AU01/01648 was filed. This is after the effective filing date of all pending claims.

Conclusion

12. Claims 42 - 55 and 60 – 61 are rejected.

13. Claims 62 – 64 are objected to as being dependent upon a rejected base claim, but would be allowable if rewritten in independent form including all of the limitations of the base claim and any intervening claims.

14. Any inquiry concerning this communication or earlier communications from the examiner should be directed to DANIEL KOLKER whose telephone number is (571)272-3181. The examiner can normally be reached on Mon - Fri 8:30AM - 5:00PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffrey Stucker can be reached on (571) 272-0911. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Daniel E. Kolker, Ph.D./
Patent Examiner, Art Unit 1649
May 2, 2008